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Acute mastoiditis

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ACUTE
MASTOIDITIS

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I Introduction

A thesis upon the subject of "Acute Mastoiditis" of necessity must be predicated upon a review of the antecedent phenomenon--acute otitis media in particular--likewise the potential complications, the expectant control and the probable pathological phenomenon concerned.

Even though the story in its entirety is of primary interest to the otologist, the general practitioner must be concerned and should be conversant with the possibilities since he not infrequently is the first consultant and delay or misjudgment on his part can react unfavorably to the patient and to the doctor.

When determining when acute suppurative otitis media passes into the surgical stage of mastoiditis, one cannot draw a line of demarcation. It is a recognized fact that in one case it may take weeks before the mastoidal symptoms develop, while in another, such symptoms appear several days after the onset of the tympanic infection.

There are symptoms common to both; some of the symptoms of otitis media may gradually progress into those of mastoiditis and institute the appearance of a

new group of symptoms which clearly indicate that the disease has advanced into the stage of mastoid involvement.

II Classification of Acute Mastoiditis

Many classifications have been advocated and probably not any one entirely fills every requirement. Kopetzky (35) divides acute mastoiditis into the coalescent and hemorrhagic types, and the coalescent into the classical or painful, painless or asymptomatic and the perforating type (subperiosteal and Bezold). Some otologists do not believe the hemorrhagic type is a distinct clinical entity but that it represents the early stages of a virulent infection in which the mucous membrane and cellular structure bleed easily.

Ersner (19) agrees with the above classification, but classifies acute mastoiditis from the symptomatic standpoint rather than the pathologic. He classifies the coalescent type of the disease into two varieties, (1) typical and (2) atypical mastoiditis. He considers the hemorrhagic type a distinct clinical entity. The writer believes this to be the best classification and will be used throughout this paper.

Ersner's classification would be as follows:

A. Coalescent type of Acute Mastoiditis

1. Typical Mastoiditis
   (a) Acute Mastoiditis with Perforations
      (1) Subperiosteal abscess
      (2) Bezold's abscess
      (3) Zygomatic Perforation
      (4) Abscess in the posterior triangle of the neck
   (b) Acute Mastoiditis with Special Neurological phenomena
2. Atypical Mastoiditis
   (a) Primary Mastoiditis
   (b) Acute Mastoiditis in Diabetes Mellitus
   (c) Mastoiditis due to Type III pneumococcus

B. Acute Hemorrhagic Mastoiditis

The acute mastoiditis with perforations has been classified with the complications by some authors, but this type will be taken up under the typical mastoiditis grouping.

III History

The history of acute mastoiditis dates back to Hippocrates (25) who first wrote on the treatment of acute inflammations of the middle ear. He wrote at length on depletion by local blood letting, steaming, the instillation of warm mild drops, a cathartic and rest. He describes the pain:

> "Acute pain of the ear, with continual and strong fever, is to be dreaded; for there is danger that a man may become delirious and die. Since, then, this a hazardous spot, one ought to pay particular attention to all these symptoms from the commencement. Younger persons die of this disease around the seventh day, or still earlier, but old persons much later; for the fevers and delerium less frequently supervene upon them, and on that account the ears previously come to a suppuration, but at these periods of life, relapses of the disease coming on generally proves fatal. Younger persons die before the ear suppurates: only if white matter run from the ear, there may be hope that a younger person will recover, provided any other favorable symptom be combined."

From this early time until the present there has been one outstanding master of otolaryngology each century making some advance in this field of medicine.

The mastoid was not operated upon until Petit (52) in 1760 successfully opened the mastoid cells to relieve suppuration. Others followed with success, but in 1791, Kolpin (33) operated upon Berger,

a celebrated Danish court physician, to relieve a severe deafness with tinnitus. Death followed from a purulent meningitis and sinus thrombosis. This incident is said to have retarded the development of mastoid surgery for nearly a century.

It was not until 1864 that Turnbull (66) in America and Mayer (48) in Germany revived the discredited operation. (The Wilde (67) incision, 1853, was not an operation upon the mastoid but a drainage for a post-aural abscess). In 1873 Schwartze and Eysell's (60) epoch making contribution laid the foundation for the modern mastoid operation.

Thies (65) in 1907, succeeded in perfecting the radical operation through the external auditory canal. In this country the endaural approach to acute and chronic supputation has been developed by Lempert (42), who has reported 1,780 operations of the mastoid and middle ear.

IV Anatomy

The true mastoid process is evolved from the petromastoid part of the temporal bone, which becomes fused with the squamo-zygomatic portion of the squamous part and the tympanic part to form the entity known as the mastoid.

Lingeman (43) gives the anatomy concerned in acute mastoiditis. First, the eustachian tube, which connects the middle ear with the throat; second, the middle ear or tympanic cavity, which contains the malleus, incus and stapes, and which has for its external wall the tympanic membrane. The upper part of the tympanic cavity is called the attic which leads through a small door called the aditus ad antrum. This rather large cavity connects posteriorally with the mastoid cells, the number and size of which vary enormously in different individuals.

Shillinger (59) reports that the middle ear and antrum from which cells develop are laid down and remain as one continuous cavity, the lining membrane of which is continuous with that of the eustachian tube and nasopharynx.

According to Black (8) the cell types of the mastoid process are: (1) pneumatic, large cell type; (2) diploetic, small cell type, and (3) sclerotic, hard type.

Kopetzky (35) describes the growth of the pneumatized bone which is the normal type of bone found in the normal ear. The diploic and the sclerotic types of bone are abnormal variations of the pneumatized bone. The middle ear, the attic, and the antrum are filled with a highly embryonal type of connective tissue at birth. This tissue is covered with a delicate layer of epithelium. Like all the other bones of the skull at birth, the mastoid process is composed of spongy bone, and has between its bony trabeculae spaces which are filled with a fatty bone marrow.

He goes on to say that when aeration ensues, and this highly embryonal tissue is subjected to the atmospheric pressure, there follows a contraction of this connective tissue. From the antrum this highly myxomatous type of connective tissue grows into the marrow spaces in adjoining the mastoid process. Normally, this ingrowth of connective tissue continues until it has replaced all the marrow cells.

As the child grows, so too does this embryonal subepithelial connective tissue begin to assume its adult form. This is finally accomplished by a process of slow fibrosis and contraction until it becomes the sub-mucosa of the middle ear. The subepithelial tissue within the mastoid process undergoes the same changes that it does in the middle ear. After it has replaced the marrow in the intratrabecular spaces, it too contracts and causes its epithelial covering to sink in after it. After this contraction has been completed, there remains only a thin layer of connective tissue covered by epithelium lining the bony trabeculae. Thus the spaces in the mastoid process which formerly contained marrow are now air-containing and communicate with the antrum and the middle ear.

According to Schillinger (59) this process of pneumatization is governed by vital and anatomic factors, the influence of which causes each mastoid to attain an individual cell pattern which differs from that of its mate and is sufficiently characteristic for actual identification.

Batson (6) states that at birth the temporal bone occupies a position on the latero-inferior surface of the skull. It is made up of three distinct parts, all

of which may be seen from the external surface, and all of which may be encountered during operation. The thin upper part is the pars squamosa. It extends down to the upper margin of the drum membrane. The annulus tympanicus is an incomplete ring continuing downward and medially from the pars squamosa. Separated from these two by definite sutures is the pars petrosa, which comprises the exposed part of the temporal bone seen behind the annulus and below the pars squamosa. These three bony parts with the membrana tympani, which is attached to the groove in the annulus, enclose the air cavity of the middle ear and its extensions.

The classification of Meltzer (49) of cell groups as to the position of the lateral sinus and relation to the cortex, digastric crest, posterior wall of the canal, etc., determines to what extent pneumatization will occur and causes a definite arrangement of cells in relation to it. Once the sinus plate is exposed and its position in relation to the structures named is noted the cellular pattern of the mastoid becomes known.

Groups.

1. Superior group.--(a) Antero-superior cells. This group includes the superficial and deep zygomatic

cells. (b) Postero-superior cells (angle, petrosal cells). This group includes the cells underlying the tegmen mastoideum and occupies the angle formed by the anterior and posterior surfaces of the temporal bone. These are the important cells extending posteriorly from the antrum to the lateral sinus where it joins the cortex of the squama. Occasionally, the cells of the angle extend into the parietal and occipital bones where they come together at this point.

2. Antero-inferior group.--(a) Cells of the posterior wall of the canal and the tip. These cells extending from the antero-inferior margin of antrum along the posterior wall of the canal include the cells at the tip as far back posteriorly as where the digastric crest joins the cortex. The cells lie above a plane passing horizontally to the facial canal. The deeper cells of the canal wall coalesce with the next group, when present. (b) Retro-facial cells. These cells lie posterior and medial to the facial canal. Superiorly, they come in contact with the deep subantral cells around the lateral and posterior semicircular canals, and inferiorly with the cells overlying the lateral sinus in the direction of the jugular bulb. The extent of development of these cells is dependent

upon the position of the lateral sinus.

3. Mesio-posterior group.--These cells are the intermediate group lying interposed and coalescing with the superior and antero-inferior groups. They make up the greater mass of cells and lie in front of, immediately over and posterior to the lateral sinus. The posterior cells are commonly designated as peripheral or marginal cells, lying above and below the emissary vein.

Williams (68) describes the auditory canal of the infant up to one year of age, as being from 16 to 18 mm. long, and the breadth being equal to that of the adult. The pharyngeal orifice is on a level with the floor of the nose and infected material consequently from the nose finds easy access to the tube. There is no angle or isthmus, and the tube is practically horizontal.

Black (8) points out that the mastoid region of the new born is flat, the process itself is absent, the antra, the digastric fossa and the stylomastoid are superficially placed. The mastoid process develops with the increase in size of the sterno-mastoid muscle. At the end of the first year, when the infant attempts to exercise its balancing power, a considerable increase in the size of the mastoid process

occurs. The normal development of air cells commences at about the age of 2 years. If pneumatization is not interfered with it is complete by the end of the fourth to fifth year.

The growth of the mastoid is practically completed at puberty but there will be an increase in thickness of the outer layer and a further extension of the cells in the vicinity of the sinus and apex.

A disease of the middle ear occurring during the first few days of life, according to Kopetzky (35), will cause a disturbance in the process of pneumatization. This may occur either by a foreign-body irritation or by infection. Taylor (62) states that a non-pneumatized mastoid in adults means otitis media in childhood. An otitis media during infancy prevents pneumatization or arrests further development when pneumatization has already started.

V Pathology and Types

The pathological changes which occur in acute middle ear suppurations resemble the changes which take place in acute inflammation of the nasal or any other mucous membrane, modified by the shape and relationship of the infected cavity. According to Dwyer (14) in all but the rarest instances the infection reaches the tympanic cavity by way of the eustachian tube. The first change that is noted in the mastoid is an intense hyperemia and swelling of the mucous membrane. This is rapidly followed by the outpouring of an exudate which is at first serous but after a short time becomes seropurulent or mucopurulent from diapedesis of leukocytes and shedding of epithelial cells. The eustachian tube takes part in the process and on account of the narrowness of the lumen at its tympanic end speedily becomes blocked.

Black (8) brings out that in the meantime the mucous membrane has become swollen to from ten to twenty times its normal thickness so that with the continually increasing exudate and the narrowed cavity there is considerable tension which causes severe pain. The tympanic membrane becomes bulged outward

and sooner or later gives way and allows the copious discharge of pus. If, however, the drum membrane does not rupture early the purulent exudate is forced back into the mastoid cells and may fill them completely. This exudate in the mastoid cells then, according to Kopetzky (37), exerts a pressure on the nutrient arteries supplying the bony inter-cellular framework. There is a resultant necrosis of the bony inter-cellular walls and a coalescence of the mastoid cells forming larger cavities which, if the disease remains unchecked, converts the entire mastoid process into one pus cavity.

Perforation of the drum is not a purely mechanical process but is preceded by small cell infiltration of its layers with destruction of part of the fibrous network.

In a large proportion of cases perforation occurs before the mastoid cells are involved to any serious extent and the discharge of pus is followed by a gradual recession of the inflammation, lessening of the discharge and finally healing of the perforation and return to normal. When there is a delay of some days before perforation occurs and the inflammation continues the mastoid cells nearly always become involved. It

is then desirable to perform myringotomy to allow the escape of pus. Statistics prove that myringotomy cuts down the incidence of complications. Krentz and Wetter (40). When the subsequent drainage through the perforation is free the inflammation in the mastoid may clear up although, on account of the complexity of their arrangement, drainage from the more distant cells must be imperfect at the best and healing is protracted in these cases.

When the drainage is insufficient or absent and the infection severe the mastoid cells become intensely inflamed, necrosis of the mucous membrane occurs, the bony framework is broken down and absorbed and an abscess is formed. In time, the pus works its way to the surface or through the inner table of the skull causing a sub-periosteal or intra-cranial abscess.

VI Pre-disposing factors

Of the direct causes of acute middle ear suppuration the common cold or coryza is by far the most frequent. It also occurs in the exanthemata such as scarlet fever, measles, diphtheria, whooping cough and in fact in any of the upper respiratory infections. In the large majority of cases the infection reaches the middle ear by way of the eustachian tube. Age has an influence on the incidence of acute middle ear suppuration, children being more subject to it than adults on account of the presence of tonsils and adenoids, much wider eustachian tubes and sinus diseases.

Kulkin (39) in writing upon the age incidence of acute mastoiditis brings out that 45 per cent is seen in children 5 years of age or younger while 80 per cent occurs in children under 10 years of age. His findings of the incidence of mastoiditis on either side was right side 49 per cent; left 41 per cent; bilaterally 10 per cent.

Kopetzky (35) brings out that a foreign body irritation, such as meconium, vomitus, or vernix caseosa entering the tympanic cavity the first few days of life will cause inflammatory reaction and cessation of pneu-

matization.

Leathart (41) therefore brings out the fact that young children should never be nursed or fed while lying on their backs. This may seem to be an insignificant point but it would be interesting to work this point out in detail.

Lynch (44) in writing upon the geographic virulence says that acute infections of the mastoid reach their peak in the spring following the cold weather, not in the summer, and do not again begin to rise until the fall, when sudden climatic changes are seen. He gathered Hemolyticus streptococcus organisms from different parts of the United States, and using the guinea pig as the lab animal, studied the innoculated animal with reference to climate, diet, exercise and illness. From his work he concluded that those areas of the country where the climate varies markedly; where extremes of cold are encountered; and where open air exercise is thus limited, over a period of time the resistance is lowered and the population is thus more susceptable to infection, this coupled with the fact that the infection gains virulence because there is less in the natural elements to combat and decrease the virulence.

Fabricant (20) writing on the climatic factor in mastoiditis states that the precipitation of disease occurs in the wake of a cold front, when the functional status of the mucous membranes of the nose and throat has changed. A few hours to one or more days may elapse before the clinical symptoms make their presence felt. It is this latent, or incubation period, that not infrequently complicates the picture, because the actual initiation of the disease follows the cold front closely.

One cold front may be passed clinically without harm. It is the superimposed cold front, striking the human organism before recovery has been possible, that results in further damage.

Campbell (11) observed the close association of sinusitis in children with purulent otitis media. He described sinusitis as the most common of all infantile diseases. He found sinus disease and acute otitis media associated in one-hundred consecutive cases.

In dealing with the contagious diseases and the otitides which follow, Williams (68) has special reference to scarlet fever and mastoiditis. The cases studied were collected between 1934 and 1937 from the Philadelphia Hospital for Contagious Diseases. Williams

is of the opinion that scarlet fever produces more acute otitis media than does any other specific disease. The second source of origin is given by him as measles.

It is interesting to note that in 1860 Toynbee (64) commented that the most frequent causes of disease in the mastoid cells, at this time, are scarlet fever, measles, small-pox, and scrofulous affections.

It is well known that in some years the organisms infecting the respiratory tract in epidemics are much more virulent than in other years. There is a distinct variation in the type, nature, severity and tendency to complications in different years and occasionally in different months of a given year. Kopetzky and Hadjopoulos (38) studied this variation over a period of six years. The report, concerned mostly with streptococcic infections, showed that they reveal orderly periodicity, with a suggestion that a major cycle embraces five to six years. They report that the height of the last cycle was in 1936 and 1937.

If we assume the above study to be correct, this would account for the varying results in the treatment that has been reported in the last 6 years.

Krentz and Wetter (40) in studying 300 cases of

acute mastoiditis at Henry Ford Hospital, found that 42.5 per cent presented frank clinical evidence of paranasal sinus disease, and commented on the fact that with x-ray the percentage might have been higher. They also found that 62.5 per cent of the 300 cases studied, had not had tonsillectomy or adenoidectomy. In 17 recurrent mastoid infections, and in all 21 cases that developed chronic otorrhea, sinus disease was definitely present. Tonsils and adenoids had not been removed in 4 cases that developed chronicity and in 21 cases that recurred.

Obviously bacterial infection serves as the fundamental cause through extension from the tympanic cavity and antrum into the contiguous mastoid cells. The resultant edema and exudation instigates the pathological chain of events which bring about the varying mastoid conditions encountered. McCaskey, Sims and Estlick (45) say that the modifying factors are essentially: (1) the kind of mastoid (whether cellular, diploic, or non-pneumatized), and (2) the type and virulence of the infecting organisms. They also bring out the fact that debilitating diseases such as anemia, chronic nephritis, diabetes, tuberculosis, and syphilis, and the acute infections, particularly scarlet fever, measles,

and other severe upper respiratory infections pave the way for mastoiditis.

In a practical sense, the chief underlying factor in the production of mastoid involvement is without doubt, the improper care of otitis media.

VII Bacteriology

The micro-organism most frequently recovered in acute inflammatory conditions of the ear is the streptococci in one or another of its varieties. Next in frequency are the pneumococci and the staphylococci in that order. Other less frequent organisms are the pneumobacillus, Bacillus pyocyaneus, diphtheria, etc. While streptococci are capable of causing severe otitis and mastoiditis, their virulence is greatly increased in healthy individuals when there has been some antecedent infectious disease.

Most of the reports are nearly the same in reporting the percentage of the micro-organisms found in cases of acute mastoiditis. Dwyer (15) in 174 cases of acute and sub-acute mastoiditis, finds the Streptococcus hemolyticus in 65 per cent; the Streptococcus mucosus capsulatus in 20 per cent; and the Staphylococcus pyogenes aureus in 8 per cent. The remaining cases showed infections with Friedlander's Bacillus, Bacillus pyocyaneus, Bacillus diphtheriae, and pneumococcus. Two organisms were isolated in 10 of the cases--Streptococcus hemolyticus with Bacillus pyocaneous; Staphylococcus pyogenes aureus with Bacillus pyocyaneus;

Streptococcus mucosus with Bacillus pyocaneous; Streptococcus mucosus with Friedlander's Bacillus, and Streptococcus hemolyticus with Bacillus diphtheriae. Dwyer also reports that very frequently there is no relationship between the organism found on culturing the discharge from the tympanic cavity, and that found in the mastoid process. He believes the finding of the Streptococcus mucosus to be the most dangerous.

Krentz and Wetter (40) in reporting 300 cases gave the following:

Streptococcus hemolyticus	65 %
Streptococcus viridans	8 %
Staphylococcus aureus	3 %
Staphylococcus albus	7 %
Pneumococcus	6 %
Type I	2 cases
Type II	2 cases
Type III	16cases
Type IV	1 case
Negative cultures	10 %

Page (50) in his series gives about the same incidence of micro-organisms found in acute mastoiditis.

Kopetzky (35) has found sterile pus in the mastoid process, and points out the part played by the

stagnation of pus there. There is a pressure on the nutrient arteries supplying the mastoid and it is easy to understand why no bacterial activity is required to cause the further unfolding of the pathologic picture. However, the invading organism causes the onset of the lesion.

According to Trofant (53) the terms Streptococcus mucous capsulatus, Pneumococcus mucosus capsularus, and Pneumococcus Type III, refer to the same organism. Although the streptococcus is Gram positive and the Friedlander Gram negative, both of these encapsulated organisms present similar cultural characteristics which may account for a similar clinical picture. A significant feature of these organisms is the production of a sticky mucoid colony on culture media, and a sticky mucoid reaction in the involved area of the patient, notable the bronchial tubes and alveoli of the lungs and the middle ear and mastoid cells.

Black (8) further substantiates the above by calling attention to the fact that the Streptococcus mucosus capsulatus of Schottmuller causes a type of otitis and mastoiditis which differs considerably from the ordinary suppurative form. This organism, he says, is now regarded by most bacteriologists as belonging to

VIII Signs and Symptoms

Under this heading will be discussed the various signs and symptoms that are found in all types of acute mastoiditis. There are certain signs and symptoms that differentiate the types of mastoiditis, but they will not be differentiated under this heading, but will be discussed fully under diagnosis and differential diagnosis.

A careful history is important in the evaluation of the signs and symptoms presented by the patient. The history will bring out the onset of the symptoms which is particularly important. Also the history will disclose the time and amount of the otorrhea, and whether or not myringotomy was done.

1. Pain.--The presence of pain is of great importance in a review of the symptomotology of acute mastoiditis. Where this symptom is present, the patient usually seeks relief, but where this sign is absent, the patient and the physician alike are often mislead and only the onset of an untoward complication makes them aware of the existing condition.

The duration of symptoms indicating mastoid involvement was observed by Kulkin (39). 85 per cent

of his cases were of less than 10 days duration, while 45 per cent of these had experienced pain for only 3 days or less. We can conclude from this observation that the pain is severe enough at the onset that the patient will present himself to the Doctor for the relief of the pain.

The patient who has been more or less comfortable may suddenly develop pain which may be deep-seated or localized on the side of the involved mastoid. Occasionally pain is distributed over other roots of the fifth nerve. The pain may be continuous or intermittent, violent or throbbing, and becomes worse at night. The nocturnal pain is associated with insomnia and restlessness.

2. Temperature.--Fever, since it may ascend and descend, is an inconstant factor but when present is an important symptom. Temperature may become normal with the institution of drainage. Absence of fever is no indication that the mastoid process is not involved.

3. General symptoms.--There may be physical unrest, lassitude, and anorexia. In children, cervical adenitis is a common occurrence. Nervous symptoms are also prone to occur, and may manifest themselves in

the form of convulsions, vomiting, vertigo, and an occasional suggestive Kernig and Babinski sign. Gastrointestinal symptoms often occur but may not necessarily be caused by mastoiditis.

4. Otorrhea.--Aural discharge is not a constant factor. When present, the quantity and quality of pus are among the evaluating factors in the diagnosis. Some patients may have a profuse and copious discharge. This is a typical finding in coalescent mastoiditis and occurs in the pneumatized mastoid where there is rapid demineralization with extensive mucous membrane and bony destruction. When the infecting organism is the Streptococcus mucosus, the discharge may be scanty, according to Richardson (54). The different types of organisms cause different types of discharge.

5. External auditory canal.--Inspection of the external auditory canal may reveal a swelling and narrowing of the lumen of the canal with a slight bulging and edema in the posterior superior wall of the osseous meatus. This is one of the most common findings in the acute coalescent mastoiditis.

6. Inspection of ear drum.--In acute mastoiditis, of the typical variety one may encounter; (1) a reddened swollen ear drum; (2) an ashen, grayish-colored ear

drum, which may be due to light reflection of the middle ear contents; or (3) a nipple-shaped ear drum, which is due to a localized ballooning of the weakest portion of the membrana tympani, produced by the purulent contents in the middle ear.

As a rule, the position of the ear drum is distorted. The degree of the distortion and the displacement depends upon the duration of the disease, the amount of pressure exerted upon the tympanum during the suppurative period and the resiliency of the ear drum, and its resistance to the pressure of the purulent contents. A typical finding is bulging in the posterior superior quadrant.

7. Tympanic perforations.--The tympanic perforations may be small, large, single, or multiple. The location of the perforation may be anywhere in the tympanic membrane but its location is of diagnostic value.

8. Postauricular and periauricular inspection.--In the normal individual, we find several postauricular sulci. An absence or a partial obliteration of these sulci is of considerable importance. The sulci are obliterated in infections of the mastoid where periostitis is present and in post-auricular edema due to a perforation of the cortex.

9. Palpation.--The normal mastoid process has roughened depressions and elevations and is covered by periosteum and tegmen. Upon palpation over the normal side one will find these roughened depressions and elevations. Normally the skin and periosteum are mobile. In a diseased mastoid, one will note on palpation, that there is an absence of these depressions and elevations, and that the derma overlying these structures is immobile. One of the outstanding signs of acute mastoiditis is sensitiveness to palpation over the mastoid cortex.

10. Deafness.--Conductive deafness is a typical finding in acute mastoiditis. There is a loss in the low-tone perception. The patient's ability to hear whispered and spoken voices is reduced.

Asherson (1) says that among the symptoms and signs upon which a diagnosis of acute mastoiditis is usually made, deafness is not one upon which much stress is placed. Yet severe deafness at the onset of an acute suppurative infection of the ear may be the predominating or even the only symptom or sign of the presence of acute and advanced mastoid infection.

Maxwell and Richter (46) express the opinion that rapid healing of the wound following mastoidectomy car-

ried a better prognosis in regard to residual hearing. Patients over 45 years of age showed the greatest post-operative loss of hearing.

11. Blood picture.--Hemograms often reveal a secondary anemia. The leukohematopoietic system discloses a leukocytosis, although in the coalescent mastoiditis the total leukocyte count is often significant.

Hume and Kahn (32) concluded that in the main the sedimentation rate is proportional to the severity of the infection, regardless of the nature of the complication. They express the opinion that it is of definite value in prognosis.

12. Transillumination.--The normal pneumatic mastoid is transilluminated light red; the diploic mastoid deep red; in the inflammatory mastoid there is less permeability of light than in the healthy mastoid, and the shadow will be a considerable distance in front of the hair border.

13. Roentgenograms.--Roentgen ray evidence of infection will be taken up under a separate chapter. It is universally accepted that x-rays are essential in the diagnosis and also in following the course of the disease.

IX Diagnosis

In discussing the diagnosis of acute mastoiditis, the outline of Ersner (19) will be followed.

Typical mastoiditis. McCaskey, Sims and Estlick (45) give the important points in the diagnosis of typical mastoiditis. (1) Profuse, pulsating aural discharge which persists unabated beyond two to three weeks after the onset of ear drainage. However, this may not be a constant factor. The pus is usually of a creamy consistency and has a slightly greenish tint. Chemically, there is an increase in the calcium content and microscopically osteoblasts and osteoclasts are found.

Asherson (2) states that in instances of acute purulent mastoiditis in which the external auditory canal is mopped clean and then fills rapidly in a short space of time, the refilling indicates that the discharge is coming from an overflow from the mastoid process. He calls this the "reservoir sign".

Other patients may have a scanty discharge of a sero-sanguineous consistency, which usually occurs in hemorrhagic mastoiditis where the Streptococcus hemolyticus is the causative organism. At other times, the discharge is slight due to obstructed drainage.

Cessation of the aural discharge does not necessarily indicate that there is an amelioration of the symptoms. On the contrary, it may signify that we are dealing with a mechanical factor; i.e., that there is an obstruction of drainage between the middle ear and the mastoid. The fact that the middle ear may be undergoing resolution is no indication that the disease in the mastoid is not progressing.

(2) Unabating pain in and tenderness over the mastoid, unfortunately manifest usually during the sleeping hours. Pain is usually manifested over the three classical points: antrum, emissary vein and tip. Occasionally when an isolated cell becomes involved, one will not elicit any tenderness over the three classical locations. However, in order to elicit any degree of tenderness where an isolated area is involved, one must palpate for small patchy thickened periosteal areas. If, on deep palpation, one notes a degree of sensitiveness to pressure, it may be inferred that there is a mastoid with remote areas of bony necrosis. The pain may be distributed over other roots of the trigeminal nerve. The pain may be continuous or intermittent, violent or throbbing. Ersner (19).

(3) Sagging of the posterior superior canal wall.

There is a swelling and narrowing of the lumen of the canal with bulging and edema. This is due to the fact that the posterior superior portion of the external auditory osseous canal is part of the anterior boundary of the mastoid antrum. Disease involving the mastoid produces a periostitis wherever periosteum is present. The osseous external auditory canal is covered by the same periosteum as the mastoid. Therefore, the narrowing of the lumen and the edema of the posterior quadrant is a pathognomonic finding in acute mastoiditis. The membrano-cartilaginous portion of the canal often becomes distorted secondary to the edema in the osseous canal. This is a constant factor when there is a perforation of the cortex with auricular displacement.

(4) Post-auricular swelling is a physical sign that is constant according to Leathart (41). It consists in the presence of a palpable gland or glands in the posterior triangle of the neck behind the sternomastoid muscle. The glands in this region are often easily palpable, but sometimes difficulty is experienced in detecting them, for they are small at first, but increase in size with the chronicity of the mastoid infection.

(5) Elevated temperature, particularly if it has

leveled off for a time earlier. When high temperature persists, it is usually of the septic variety and may be due to toxic absorption or venous phlebitis in the mastoid region, necrosis of the mastoid cells, or tissue destruction.

(6) Reduction in hearing. As was pointed out by Asherson (1) this may be the predominating symptom at the onset of the disease. There may be no reduction in hearing but the typical finding is a conductive deafness with loss in the low-tone perception. There is a diminished air conduction and a localization of the Weber sign to the affected side with no change in bone conduction. Audiometric graphs disclose a loss in the low frequencies.

(7) Systemic symptoms, such as headache, chills, lassitude and the like. Gastro-intestinal symptoms often occur but may not necessarily be caused by mastoiditis. The gastro-intestinal symptoms are mild flatulence, vomiting, diarrhea, etc. Nervous symptoms are also prone to occur, and may manifest themselves in the form of convulsions, vomiting, vertigo, and an occasional suggestive Kernig and Babinski sign.

(8) Roentgenograms. These will be taken up under a separate heading.

(9) Laboratory. A Schilling index with a shift

toward the left is the most significant. Hemograms often reveal a secondary anemia. The leukohematopoietic system discloses a leukocytosis, although in the coalescent mastoiditis the total leukocyte count is often significant. However, the polymorphonuclear leukocyte count is increased where bony necrosis is active. In the stormy or virulent cases of mastoiditis, one finds a shift to the left with an increase in the immature cells or young neutrophils, and a marked degenerative index in the polymorphonuclear leukocytes.

Acute Mastoiditis with Perforations.--These have been classified as complication of mastoiditis but will be taken up here.

Mastoid perforations occur whenever there is bone destruction. In this condition there is a break in the cortex with an escape of pus through the fistula. In infants where the bones are not fully ossified, the pus makes its escape through the squamo-mastoid suture.

Ersner (19) ponts out that perforations in the mastoid cortex may be due to localized halisteresis, increased localized intra-mastoid pressure, and large tip cells where the bony plate is very thin. The mastoid tip may be thin at the inner medial wall, and the

pus escapes into the digastric fossa resulting in a Bezold's (7) abscess; or the suppurative process may break through the outer wall of the tip at the attachment of the sterno-mastoid muscle, resulting in an abscess in the posterior triangle of the neck.

A perforation occurring through the lateral wall will develop into a localized subperiosteal abscess. A perforation through the anterior wall will empty itself through the external auditory canal. An edema occurring in the antero-superior auricular region is due to a perforation in the zygoma. Downward perforations empty themselves either inwardly through the digastric fossa or through the outer portion of the mastoid tip at the sterno-mastoid muscle.

Characteristic deformities may be produced by: (1) subperiosteal abscess; (2) Bezold's abscess; (3) zygomatic perforation; (4) abscess in the posterior triangle of the neck.

1. Subperiosteal abscess.--There are certain auricular displacements and changes in the outline of the soft parts about the ear which lead one to suspect mastoiditis with a perforation. In adults, an auricular displacement is infrequent, but in children it is a rather common occurrence. This characteristic

deformity is produced when a subperiosteal abscess is present.

The abscess is the result of a perforation in the mastoid cortex. The pus makes its escape through the perforation and elevates the periosteum from the surrounding bone.

Diagnostic features: If the perforation is behind the auricular attachment, the auricle is pushed forward and protrudes rather prominently from the side of the head. If the perforation is at the higher level, the auricle is pushed forward and outwards. If the perforation is at a still higher level, the auricle is displaced both forward and downward.

2. Bezold's abscess.--Bezold's (7) abscess occurs in cases where the tip cells are rather large, and the bony plate forming the medial wall of the tip is quite thin. The pus follows the line of least resistance and thus makes its escape through the lower portion of the mastoid into the neck, through the digastric fossa beneath the sterno-mastoid muscle. The pus may also confine itself between the layers of the innermost cervical fascia.

Signs and symptoms: A Bezold's abscess is usually accompanied by high temperature, although afebrile cases

are not uncommon. One observes a large mass extending from the tip of the mastoid along the angle of the jaw underneath the inferior maxilla.

Facial palsy is frequently concomitant with Bezold's abscess. This is because there is pressure upon the facial nerve in its exit from the stylo-mastoid foramen. There is a sagging of the posterior superior canal wall with a perforation, and profuse discharge. Postauricular examination reveals an obliteration of the sulci. Tenderness is most marked at the inner portion of the mastoid tip.

3. Zygomatic perforation.--The first indication of a zygomatic perforation is swelling over the temporal muscle around the zygomatic region which gradually moves downward anteriorly opposite the tragus. The swelling often resembles that of mumps, and it is not uncommon to find this type of perforation in children. The temperature behaves like the typical septic temperature, ranging from 99 to 103°.

4. Abscess in the posterior triangle of the neck.--This shows that the suppurative process has broken through the outer wall at the tip at the attachment of the sterno-mastoid muscle.

Acute Mastoiditis with Special Neurological Phen-

omena.--Facial palsy. Clinically, facial paralysis is divided into the central and peripheral varieties. Ersner (19). The former is produced by a lesion of the neurones and axones above the facial nucleus in the pons, while in the latter the peripheral paralysis is due to a lesion of the nucleus or the nerve distal to it. In mastoiditis, facial paralysis is of the peripheral type. The facial paralysis is sudden in onset. It occurs during the acute stage as the result of ulceration and caries of the wall of the canal. It may also be due to a toxic neuritis, or to the entrance of infection along the nerve branches that make their exit from the facial canal within the mastoid bone.

Gradenigro's symptom-complex: The clinical picture of this classical syndrome constitutes a triad of symptoms: (1) otitic suppuration, (2) abducens palsy, and (3) trigeminal pain. The diagnosis of the abducens nerve palsy can be easily made because the abducens is purely a motor nerve, and it supplies only one muscle, the external rectus, and its palsy produces internal strabismus and diplopia.

Recurrent Mastoiditis.--In recurrent mastoiditis we are confronted by two types: (1) recurrent mastoid infection where resolution has taken place without the

institution of surgery; and (2) recurrent mastoiditis following a mastoidectomy.

In the first type we have a repetition at frequent intervals of otitis media, associated with suppuration within the mastoid. Each attack is accompanied by profuse aural suppuration and the clinical narrowing of the canal, mastoid tenderness, etc. Within the mastoid cavity there is destruction of bone, demineralization, necrosis of cells, and, at times, exposure of such vital structures as the lateral sinus, dura, and labyrinth.

In the second type, that is,recurrent mastoiditis following mastoidectomy, we have a recurrence of all the signs and symptoms of an ordinary or typical case of acute mastoiditis. The symptoms begin with those of acute otitis media, associated with temperature, pain and tenderness, and drainage resulting from spontaneous rupture or myringotomy.

According to Kulkin (39) the number of years since previous mastoidectomy in cases of acute recurrent mastoiditis is greatest in the first three years following surgery. 25 per cent recurred within the first year; 29 per cent recurred from the first to the second year and 15 per cent in the second to the

third year. From the third to the fifteenth year, the percentage of recurrent mastoiditis following mastoidectomy was 29 per cent.

Atypical mastoiditis.--In atypical mastoiditis there is an absence of most of the classical symptoms. For example, it is not uncommon to find that temperature, pain, and aural suppuration are absent.

Under atypical mastoiditis will be considered primary mastoiditis, mastoiditis in diabetics, and mastoiditis due to Streptococcus mucosus (Type III pneumococcus).

Primary mastoiditis.--This is really a misnomer because it really is a pathologic entity secondary to tympanic involvement. In this type of mastoid infection, according to Ersner (19) the otitic picture may be absent or may have been completely overlooked because of its mild or fleeting symptoms. This type of infection may be divided into three classifications: (1) where there is postauricular edema and auricular displacement without otitic symptoms; (2) where there are mild middle-ear symptoms without mastoid phenomena, and (3) where there are fulminating toxic symptoms, the focus of the infection being in the mastoid, but without symptoms referable to the ear.

In the first type the history does not reveal an otitis media. We can assume that the symptoms of the otitis media were so mild and fleeting that they passed unnoticed by the patient. The tympanic examination is usually negative and there are no changes in the normal landmarks. The middle ear infection subsides while the mastoid destruction continues and the first inkling one receives of the presence of otitic disease is the discovery of postauricular edema.

In the second type, the patient may complain of a dull, vague, indefinite, and inconstant pain in the middle ear. Upon inspection and palpation over the mastoid, no definite tenderness is elicited, but on deep palpation one notes localized areas of tenderness. The ear drum is lusterless and opaque, and at times a fullness is found in the posterior superior meatal wall. The latter is the only positive finding which leads us in making the diagnosis of this condition.

In the third type where there are fulminating toxic symptoms, the patient presents symptoms consisting of temperature, headache, and general septic phenomena, without any symptoms referable to the ear.

Acute Mastoiditis in Diabetes Mellitus.--Acute mastoiditis in diabetics belongs to the atypical group

because in most instances there is an absence of pain and temperature. The only outstanding symptom is profuse suppuration. Some clinicians explain the absence of pain as a result of rapid halisteresis and to anesthesia of the sensory terminal nerve endings.

Diabetics are often hard to control during an infection, and the patient will show an increase in blood sugar due to the infection. Symptoms of acidosis may occur and at times becloud the otitic clinical picture. In addition, the patient may have an impairment in hearing which appears early and is of the conductive type. As the mastoid destruction proceeds, the deafness becomes more marked. Some cases show early manifestations of perceptive deafness.

Mastoiditis Due to Type III Pneumococcus.--Clinically these cases are characterized by an insidious onset, with very little, if any, temperature. One is inclined to minimize the seriousness of the otitic condition because the early symptoms are vague and mild.

On otitic examination the drum membrane may be red and bulging in the acute stage, but as it passes into the latent stage, there is a complete loss of the normal landmarks. The ear drum is lusterless and there is slight edema which shows no improvement despite re-

peated myringotomies and the institution of drainage. The color is sometimes of a dark hue and in other instances is pale. The discharge is scanty and mucoid.

Diagnosis of this type of infection is generally confirmed by smear, culture, and typing. A special thionin stain distinguishes this form of capsulated organism.

Acute hemorrhagic Mastoiditis.--Acute hemorrhagic mastoiditis is a distinct clinical and pathologic entity. This was confirmed by Pastore and Hempstead (51). They found at operation a purely hemorrhagic state and they reported no evidence of changing into the coalescent type.

The symptom of sepsis is present from the onset and is ushered in with chills, high temperature, marked prostration, and extreme restlessness. This type of infection may follow the exanthemata, such as scarlet fever, or may be due to upper respiratory tract infections, virulent tonsillitis, or other factors. At times the toxemia is so overwhelming that the otitic picture is completely overlooked.

In infants and children characteristic symptoms are extreme illness, gastro-intestinal disturbances, high temperature and often meningeal symptoms which

appear early and are by no means uncommon.

In hemorrhagic mastoiditis there are two distinct classical pathognomonic findings when myringotomy is performed; (1) myringotomy reveals the escape of a small amount of thin sero-sanguineous fluid. The discharge remains scanty and is of a sero-sanguineous consistency throughout the course of the infection; (2) there is a profuse bleeding following myringotomy, which is often difficult to control. Kopetzky (35).

A characteristic symptom is high temperature which is persistent and does not subside with the institution of drainage. On palpation tenderness is elicited over the mastoid antrum, emissary vein and over the tip of the mastoid. Laboratory findings may reveal a positive blood culture, and the Streptococcus hemolyticus is the organism prevalently found.

X Differential Diagnosis

Kopetzky (37) points out that the painless or asymptomatic type of acute coalescent mastoiditis must be differentiated from cases of uncomplicated acute purulent otitis media. It must always be remembered than any involvement of the tympanic cavity will result in a definite variation from the normal status of the mastoid cellular structure.

The painless character of this type of mastoiditis depends upon the invading micro-organism to some degree. Infections wherein the Friedlander bacillus is the causative agent come under this group, and diabetics, whatever the invading organism may be, will present this type when they develop a mastoiditis. This is due to the anesthesia which the diabetics produce in the nerve endings. Kopetzky (37) has seen such cases run their course without surgical intervention until pressure over the mastoid area revealed the dehiscent cortex and the overlying skin and the periosteum could be indented.

In contradistinction to the coalescent type, where the intercellular walls are broken down, in the hemorrhagic type they are intact. Here the mucous

membrane is thickened and swollen, but different from the gelatinous edematous swelling noted in the coalescent mastoiditis. Here the mucosa is reddened and bleeds at touch.

External otitis with furunculosis must be ruled out. This distinction usually is not difficult unless both conditions are concomitant. McCaskey, Sims and Estlick (45) say that in such cases the wisest course is to treat both and continue observation until the diagnosis is definite.

Ersner (19) gives the differential diagnosis between mastoiditis with perforations and furunculosis. In mastoiditis with perforations there is no pain on manipulation of the auricle, increased temperature, bulging of the ear drum, a profuse suppuration and positive pathological changes seen by x-rays.

In furunculosis there is pain upon manipulation of the auricle, the pain is pre-auricular and the pain is present on mastication. There is no increase in temperature, no bulging of the ear drum, the suppuration is scanty, and the roentgenograms are negative.

Williams (69) brings out the differential diagnosis between acute infections of the upper respiratory tract and acute infectious diseases such as

scarlet fever and measles.

In the first form the symptoms develop rapidly and accompany the invasion of the upper respiratory tract. The pain is usually severe at first and the drum is red and bulging. The discharge at first is sero-sanguineous, but may become purulent inside 48 hours.

In the second form, there is some pain with a slow rise in temperature. It usually follows two to six weeks after the onset of the disease especially the scarlet fever. This type is usually the less virulent of the two types.

XI Roentgenograms

Johnson (29) states that a thorough roentgenologic examination in search of mastoid disease calls for careful and ingenious technic, since not only the mastoid process but the squamous and petrous portions of the temporal bone may be involved.

He uses four projections in routine mastoid examination. (1) Standard oblique lateral projection; (2) Oblique posterior anterior projection; (3) the fronto-occipital projection and (4) an axial projection of the base of the skull in the mento-vertical direction.

Mastoid cellularity varies through a wide range from the complete absence of recognizable cells to extensive pneumatization of all portions of the temporal bone.

A completely acellular mastoid is easily recognized by its great relative density and its contracted appearance. When the opposite mastoid is normally pneumatized these features seem accentuated. Johnson (29). The mastoid process is under developed while the groove for the sigmoid sinus usually is prominent and in close proximity to the posterior wall of the auditory canal. The absence of mastoid cells,

when caused by chronic infalmmation is associated with bone sclerosis.

According to Ersner (19) the roentgen ray will disclose the following:

1. The type of mastoid, whether pneumatic, diploic, or sclerotic.

2. The topographical and regional anatomy of the mastoid, its cellular distribution, the type of cells, the position of the sinus and the surrounding structures.

3. Cavity formation.

4. Progress of the disease within the mastoid cavity, as in coalescent and recurrent mastoiditis.

5. Bony absorption or demineralization, with thinning of the outline of the cellular walls, and the gradual disappearance of the inter-cellular structure.

The roentgenologic signs of diseased mastoid cells are well known. Johnson (29) states that early suppuration results in simple clouding, to be followed in more advanced stages by recognizable absorption of cell walls. If untreated, the process may progress to the formation of large bone abscesses. If the infection subsides, varying degrees of bone regeneration will occur during the process of repair, causing increased thickness and density of cell walls.

Ersner (19) explains it more fully. He says that in the acute mastoid of recent origin there is at first hyperemia, and the congestion is the same as in any part of the body. Roentgenologic studies of both mastoids reveal that the normal side is clear and that the cellular components stand out sharply, with distinct cellular septa, while in the congested side all the normal mastoid elements are present, but in addition there is a slight haziness, appearing as though there were a slight error in focusing.

In this stage either resolution takes place or the pathology continues. We may assume that the bacteria here are active and that the mucous membranes lining the mastoid cells are becoming more edematous. We also have a constant infiltration of round cells and an increase in the leukocytes and the congested area gradually becomes purulent. The roentgenograms at this time will reveal marked cloudiness, with a tendency to obliteration of the cellular spaces which appear as if they were filled almost to the brim. The cellular septa are still visible as a minute network of fibrillated strands, and our interpretation of the plates at this stage should be that of exudation present. With the advent of an increase in the

purulent consistency there is also an increase in the intracellular pressure. The picture now presents a distinct opacity, with a blurring of the cellular septa.

Absorption, liquefaction, and cavity formation appear on the roentgenogram as dark spots or shadows more or less regularly outlined, with the edges blurred and no trabeculae visible. This is in contra-distinction to the appearance of the normal large cells which are irregular in shape with their edges sharply outlined; and the cellular septa are distinctly visible.

Brownell and Hauser (10) ran a series of 100 cases in which roentgenograms were taken before operation. They concluded from this study that the roentgenogram is never entirely negative when a mastoid disease is found surgically and probably is never clouded when the mastoid process is found to be normal at operation. They say that the roentgenogram is 80 per cent efficient in indicating the presence of bone destruction.

According to Johnson (29) it is wise to re-examine the mastoid after surgical intervention to provide graphic evidence regarding the completeness of the operation and the location and status of surgically

inaccessible cells which may come under suspicion at a later date.

Johnson (30) says that a series of x-ray films must follow the administration of sulfanilamide. The patient may feel fine and his symptoms be relieved by the use of the drug, but after discontinuing the drug the bone destruction proceeds, just as though no drug had been administered.

Pneumatized bone produces a lesion designated as osteitis. This is the only type of bone which can undergo coalescence. Diploic bone generally produces an osteomyelitis. Sclerosed bone is always found with chronicity, but is not the result of a chronic lesion. An acute coalescent mastoiditis cannot occur in sclerosed bone. Kopetzky (35).

Roentgen-ray examination plays the important part in the examination. The roentgenogram, while giving valuable data in estimating indications for operation, is used to familiarize the clinician with indication as to the type of bone upon which the lesion is based. He can then prognosticate to the degree of certainty the course that his case will take and the complications that are potentially possible with the type of bone structure the temporal bone of a given case presents.

XII Treatment

Under this heading will be discussed (1) myringotomy; (2) sulfonamide therapy; (3) resolution without surgical interference, which will also include the medical management when surgery is refused; (4) x-ray treatment and (5) surgery, with both the pre-operative and the post-operative care.

1. Myringotomy.--Williams (70) says that of all the operative otologic procedures, this is the one most commonly employed not only by the otologist, but also by general practitioner and pediatricians. The object of this surgical measure is to provide drainage for the accumulation of pus within the tympanic cavity.

According to Krentz and Wetter (40) all conditions were more favorable when the drum was incised rather than being allowed to rupture. In their series of 300 cases, with surgical incision of drum the average aural discharge lasted only 15 days and the post-operative care was 34 days.

When the drum was allowed to rupture spontaneously the aural discharge on the average lasted 30 days while the post-operative care was 39 days.

They concluded that besides the relief from pain, myringotomy has a distinct tendency to prevent the development of surgical mastoiditis from otitis associated with scarlet fever and measles.

2. Sulfonamide therapy.--The drug, sulfanilamide, has been the most widely used drug of this group. It is still a little too early to evaluate this drug correctly because there are so many conflicting reports. In the main however, it is generally believed that sulfanilamide has a definite place in otology. The drug was first used in mastoiditis in 1937, and Horan and French (27) reported in 1938 that during the last half of 1937 and the first half of 1938 the routine use of sulfanilamide reduced the ratio of acute mastoiditis to otitis media from 22.7 per cent to 4.5 per cent. As with all new drugs, sulfanilamide enjoyed great popularity at first, but since then the drug has been studied more thoroughly and evaluated more correctly.

According to Baker and Bradford (5), the person with toxic symptoms, high temperature and mastoid tenderness, with a history of a discharging ear of from a few days to a week's duration, give the best response to sulfanilamide therapy. In their cases the

temperature would frequently become normal within twenty-four hours, and the toxic symptoms as well as mastoid tenderness would disappear. Occasionally the middle ear would become dry within a few days, but more often the ear continued to discharge for a longer period, while the patient remained free of symptoms.

The development of fever in patients receiving sulfanilamide was not uncommon. It usually occurred after the drug had been administered for several days.

Fenton (21) points out that the use of sulfanilamide has special value in the management of acute streptococcic invasion of the middle ear and mastoid. Streptococcic infections of the eustachian tube leads very swiftly to its closure and the rapid multiplication of these germs in the middle ear. He brings out that all are familiar with the picture of the "red-hot" ear with bulged and scarlet drum, which fills so rapidly with toxic fluid that the tympanic membrane does not have time to soften and break as in milder infections, hence favoring the rapid spread of the infection to the mastoid cells, the sigmoid sinus, or even through the tegmen tympani to the dura. Twelve to twenty-

four hours is ample time for these infections to spread through most of the air cells communicating with the middle ear. Sulfanilamide is not a substitute for myringotomy, which must be done early and repeated whenever necessary. But one finds this drug invaluabe as an aid in disease of the temporal bone due to streptococci and pneumococci. He states that there is apparently no antiseptic or sterilizing action by the drug; it does not kill organisms, but seems either to neutralize or decrease the amount of their toxins, or at least to facilitate the production of anti-bodies called out by such toxins present in the blood stream and tissues.

Kopetzky (36) in 1938 expressed the opinion that sulfanilamide will clear bodily fluids infected by streptococci, but it does not seem to kill streptococcus located in an active osseous lesion.

In 1940 Rosen (55) reported a case in which the using of sulfanilamide masked a case of acute mastoiditis. The patient received 40 grains a day for seven consecutive days. During this time the pain in the ear gradually disappeared, the discharge diminished and the patient could hear a soft whisper. On the

eighth day, because of toxic manifestations of headache, vertigo, and a rise in temperature, sulfanilamide was discontinued. The next three days saw a gradual subsidence of the toxic manifestations, but on the fourth day there was a recurrence of severe pain and marked mastoid tenderness. The discharge increased.

A thorough simple mastoidectomy was therefore performed. Pus was encountered as the cortex was removed. There was an epidural abscess in relation to the antrum. The entire mastoid was the seat of extensive purulent bony necrosis.

Hebble (24) in a quantitative study of 17 cases, came to the conclusion that his patients showed a generally poor response to conservative medical treatment, including the use of sulfanilamide. However, if the patient's ear has been discharging less than 4 weeks, continuous sulfanilamide therapy throughout several weeks seems advisable providing there are no definite toxic effects. However, if the otorrhea has already persisted over 4 weeks, when the patient is first seen, surgery seems feasible. He also brings out the fact that sulfanilamide does mask certain typical symptoms in intra-cranial involvement, but this is not a contra-indication to the use of the drug.

Kopetzky (37) writing in 1939 of his own experiences, states that there were less patients coming to surgery with the mastoid in a condition to require surgical intervention, was less in 1937, before sulfanilamide was given in almost every case, than during the winter of 1938 and 1939, even though sulfanilamide was used much more frequently. He attributes this to cycles in which there is much more mastoiditis than in other years. These facts would have to be considered in the true evaluation of the drug. As previously stated, it is too early to know the true effect of the drug in every case of mastoiditis.

There is much debate on the dosage of the drug. The accepted dosage of sulfanilamide for infants and children in acute mastoiditis is given as 10 grains every 4 hours for the first 24 hours, and 5 grains every 3 hours afterwards. The concentration in the blood and the spinal fluid should determine the amount given. The concentration in the blood and spinal fluid should be kept at 9 to 11 milligrams / 100 cc.

Sulfapyridine has not been used as extensively as the other drugs in the sulfonamide group. Richardson (54) used this drug successfully in the treatment of a case of mastoiditis caused by the Streptococcus

mucosus. The disease ran the irregular course characteristic of this organism, with scanty discharge from the right and absence of discharge from the left ear. In the latent stage approximately two months, deafness and tinnitus were the only symptoms. In the first instance both ears appeared to be equally involved. Under treatment with sulfapyridine the right ear cleared up completely after two and a half months, but the left ear required operative intervention. Although the operation revealed extensive destruction of bone in the left mastoid, with considerable exposure of the dura mater, no complications ensued. The author called attention to the fact that this was due to the protective influence exerted by the sulfapyridine.

To sum up the situation so far, Curry (12) in 1941 says that the use of sulfanilamide and other chemotherapeutic agents has modified the treatment of otitis media and its complications. These drugs have been sensational in the successful treatment of hemolytic streptococci septicemia and meningitis, but their use has also masked serious intracranial complications. It should be emphasized that the administration of sulfanilamide may not influence the healing of an ab-

scess in the mastoid cells.

3. Resolution without surgical interference.-- It has been shown that the hemorrhagic type of mastoiditis never ends in resolution. Kopetzky (35) states that because of the rapidly developing complicating lesions the patient is subjected to operation. Therefore, the discussion of resolution without surgical intervention must of necessity be limited to the coalescent type of mastoiditis which gets well with the institution of adequate drainage from the middle ear, rest in bed, catharsis, light diet and local treatment.

Atkinson (3) has shown that as soon as an avenue for escape of pus has been provided in the mastoid process, bone destruction ceases. The coalesced mastoid cells containing granulation tissue cannot reform into a series of cells with epithelium-lined walls, but part of the granulation tissue does become absorbed and the remainder forms firm connective tissue. In an endeavor to encapsulate the necrotic process, new bone is formed from the surrounding area. Where the cortex has been perforated there is a regeneration of bone from the inner surface of the periosteum.

We may consider a mastoiditis as resolved when

all constitutional and local symptoms have disappeared, the middle ear has become dry, the perforation in the membrana tympani has closed over and the hearing has returned to normal. Where there were repeated incisions of the membrana tympani with a resultant deformed drum head, the hearing acuity is permanently diminished.

4. X-ray treatment of mastoiditis.--Dysart (17) calls attention to the fact that improvement was noted in acute mastoiditis following the taking of x-ray pictures for diagnosis. In reporting a series of cases he came to the conclusion that small doses are better than large doses, as too large doses can aggravate the process instead of reducing it. In his cases, x-ray treatment was started as soon as the patient presented symptoms of mastoid involvement.

Schillinger (58) describes the progress after exposure of the infected mastoid to the roentgen ray. The temperature drops rapidly to normal, and there is an absence of pain and insomnia. There is also a diminution in the amount of discharge and a change in the character of the discharge from purulent to mucopurulent. His conclusions are that by the use of irridiation on cases of acute mastoiditis in which

operation is indicated will, in a large majority, cause resolution and cure without operation.

According to Ross (56) the invasion of the mastoid cells had stopped after x-ray therapy because of the inhibition of the multiplication and motility of the bacteria. The increased phagocytosis of the bacteria present was indicated by the increased thickness and amount of discharge during the first 2 days of treatment; also by the subsequent thinning and lessened amount of the exudate from day to day until all bacteria present had been phagocytosed. At this time all discharge from the ear stopped. This was in a series of 47 cases, and in all the cases observer, the pain was noticeable less following each treatment, and it was usually relieved entirely after the fifth treatment. All discharge from the ear stopped in from 10 to 14 days in his series of cases.

Writing in 1932, Ross (57) gave a report of 41 cases during 17 years that he had treated. These cases all recovered without operation.

Dysart (17) gives the picture dose as being not more than 5 or 10 roentgens. The average dose measured by "r" for early cases of mastoiditis is considered to be 50 or 60 r; for the more chronic cases, doses

as high as 100 r or more are used. Measured by the erythema method, 25 to 50 per cent of an erythema dose has been advised in early acute cases, and as high as 80 per cent of an erythema dose for the chronic cases. (Erythema dose is that quantity of radiation which, when delivered at one sitting will produce in at least 95 per cent of cases a definite reddening of the skin within one week, followed by bronzing, without permanent injury to the skin. An erythema dose is equal to 350 r unfiltered, while it is equal to about 450 r if filtered with 4 millimeters of aluminum).

5. Surgical treatment of mastoiditis.--The pre-operative management will be taken up first.

There is no hard and fast rule for treatment previous to operation. One must be guided by the course of the disease. Conservatism should be the guide during the first two to three weeks. Allay the fears of the parents and explain to them the disease itself, its course and complications.

Each operator has his own particular pre-operative care so the routine of Black (8) will be described. The ear should be cleansed with small cotton applicator pledgets, never by douching the ear. A

well balanced diet should be fortified by small amounts of concentrated vitamins each day; mild laxative should be given when necessary, and fluids, orange juice, etc. A daily four time temperature chart should be kept; small doses of aspirin T. I. D. for pain, temperature and nervousness, and small amounts of barbital of some kind for sleep if restless, should be given.

McCaskey, Sims and Estlick (45) add intravenous fluids, blood transfusions, and sulfanilamide when streptococcus hemolyticus is cultured from the aural drainage. They call attention to the fact that mastoidectomy is not an emergency operation, except in cases with complications or other rare occasions. Therefore, it is necessary to get the patient in the best possible condition for the operation.

The patient is prepared for general anesthesia, or more rarely for local analgesia. The hair around the affected ear is generously clipped and shaved, and, when complications are anticipated, a wide area of the head and neck is prepared. The remaining hair is held back by a gauze skull cap, the edges of which are collodionized. The field of surgery and ear are scrubbed with ether and painted with tincture of merthiolate and covered with sterile dressings the night

before surgery.

Surgery of the mastoid.--There are two methods of mastoidectomies. The one of most popular use is the post-auricular approach. Lempert (42) has described an endaural, antauricular approach and this latter operation will be described later.

The fundamental purpose of the operation is to eradicate the disease located between the inner and the outer tables of the temporal bone--the mastoid process. The operation is not completed until the removal of all diseased areas areas has been accomplished. The entire contents of the mastoid process are to be eviscerated.

The post-auricular mastoidectomy has become a standardized procedure and the variations as to the operation are with the operators. Williams (70) says to "learn one technique well and then add your own variations to suit your own ideas." The fundamentals of the operation will be given as described by McCaskey, Sims and Estlick (45) and Black (8).

The mastoid operation is divided into the simple and radical operations. The simple mastoidectomy is incision and drainage with removal of all diseased bone. The radical mastoidectomy is the same as the simple with

addition of removal of middle ear contacts and tympanic membrane. The stapes is left in place, the facial ridge is lowered and the bridge taken down.

The patient is placed on the table with the head turned on the healthy side and the tip of the mastoid somewhat elevated. The head is supported by sand bags and the mastoid is again prepared for surgery with ether and tincture of merthiolate. Sterile head drapes and towels are placed over all but the operative field and a special mastoid sheet with aperture cut out is placed over these. This sheet is supported upward at one corner by a standard thus making a tent behind which the anesthetist works.

Incision should be made posteriorly far enough to just circle the outer limits of the process from the tip below to the level of the zygoma. In this manner there is a wide flap; this allows the operator to retract forward and not disturb the periosteum posteriorly. After hemostasis the periosteum is raised and retracted until the supramental angle and the spine of Henle are identified. The tip fiber of the sternomastoid is divided with sharp pointed scissors. In very young children with the mastoid not developed the incision should be made somewhat higher to avoid the facial nerve at the tip.

A large gouge is used in the exposure of mastoid cells. The cortex is taken off first paralled to the posterior canal from the top of the suprameatal triangle to the tip. This groove is deepened along its length until the mastoid antrum is exposed and the horizontal canal identified. The rest of the cortex is removed and the cells are obliterated in the following order: Superior cell group, posterior superior cell group, postantral cells; the outer antral wall and the upper and outer aditus are enlarged and the zygomatic group, the mesioposterior group, the anterior inferior and the retrofacial and cells between the sinus and the bulb. The posterior canal wall should be taken down and all the pockets and overhang should be obliterated and the edges smoothed down. This eliminates tender areas if scar tissue formation occurs, and makes the auricle fit into the mastoid would without deformity.

According to Smith (51) the chief fault with the average operator is the failure to properly lower the facial ridge. This is best accomplished by using a sharp Richards curette to gently shave away the bone over the course of the mastoid segment of the facial nerve from the horizontal semicircular canal down to a line parallel to the level of the floor

of the middle ear.

All of the diseased bone in the mastoid should be removed to healthy plate both in the middle and posterior fossae, the dense bone forming the external wall of the attic must be removed to the thin dural plate forming the floor of the cavity. The annulus is completely removed along with the granulations in the eustachian tube and middle ear; then the cells in the region of the tube and the hypotympanic space are exenterated.

The wound must be entirely free from fresh or clotted blood; after the wound is thoroughly cleansed with peroxide and salt solution and completely dried.

The amount and kind of drains varies with the individual, but the writer believes the method proposed by Smith (51) to be the most feasible. The wound is then lined with strips of rubber tissue, the type used in making cigarette drains, and packed with iodoform gauze. The periosteum and temporal fascia are sewed back in their original position with interrupted catgut sutures.

He points out that the advantages of the rubber tissue are three-fold. It avoids the very painful first dressing so bitterly complained of by the pat-

ient. The rubber tissue prevents a gauze from adhering so that both may be removed without pain: finally the rubber tissue avoids the new granulations which become entwined in the meshes of the gauze when it is placed in the bare bone, this in turn minimizes the bleeding.

Michel clips are used to close the wound, 2 or 3 clips being adequate. Jones (31). The skin clips are the method of choice but black silk and in some cases subcuticular plain catgut No. 0 is used.

Sirus drain may be placed with one end in the antrum cavity and the other extending through the lower extremity of the wound, protruding for about one half inch. A plain drain moistened with alcohol and boric acid, equal parts, may be packed in the external canal.

Post-operative.--The first dressing.--Smith (51) stresses the fact that the after-treatment should begin with a cavity free from new or clotted blood. Where the bleeding persists a pledget of 10 per cent cocain is used and a small piece of plain gauze moistened with 95 per cent alcohol is placed in the cavity for twenty-four hours, the packing discontinued as soon as the bleeding is controlled.

Irrigations are not used unless there is a second-

ary purulent infection present, then boric irrigations are used three times daily, followed by ten drops of 95 per cent alcohol. If the alcohol drops are too painful, a few drops of 10 per cent cocain solution used shortly before the dressings will relieve most of the pain. Alcohol is one of the best deterrants to unwanted granulations.

The wound is dressed with the drain in place. The use of glass and hard rubber tubes have been largely discarded according to Tobey (63) as drains. There is much variation in the time that the first dressing is left in place. Jones (31) states that the outer dressing and drain in the canal are to be changed daily without disturbing the clips or drain. On the fourth or fifth post-operative day, the clips are removed and the Sirus drain changed for one of a smaller size.

McCaskey, Sims and Estlick (45) say that the dressing should not be changed until the third post-operative day unless the drainage saturates the dressing.

All the authors agree that the number of days elapsing between dressings depend upon the amount of the discharge. A few need to be dressed daily,

but more commonly an interval of one to three days is preferable.

Sometimes a wound must be left open. When this is necessary a secondary closure is done some days later and the after care following the closure is the same as has been described.

Statistics indicate that there are fewer complications when mastoid operations are performed during the second and third weeks of the course of the mastoid infection. Guthrie (22). The primary reason for doing the radical operation is to save the patients life, and when imperative indications for the radical mastoidectomy exist, there seems to be no difference in opinion as to the advisability of immediate operation.

Duration of the hospitalization is usually around 8 to 12 days in the uncomplicated cases.

From time to time some untoward sequel, such as an unusually depressed scar, the persistence of a small fistula, the formation of a keloid, or recurrent pain in the incision, has made one wonder whether it would be possible to approach the mastoid satisfactorily by some route other than the postauricular. For many years operations through the external auditory

meatus have been suggested and performed, and their range has been some what extended since the introduction of efficient local anesthesia: but they appear to have been used in, and considered applicable to, special cases only.

The most popular operation of this sort is the one described by Lempert (42). His operation consists of three stages. Stage 1: operation in the superficial soft parts covering the temporal bone. Stage 2: operation on the temporal bone proper. Stage 3: surgical treatment and guidance of the membranous and osseous wounds resulting from the first two stages.

Stage 1 consists of the creation of a mobile membranous and extracartilaginous window within the external auditory canal for endaural, antauricular surgical approach to any desired part of the temporal bone.

The endaural, antauricular operation on the temporal bone is not performed through the external auditory canal but through a widely opened mobile membranous and extracartilaginous endaural window which, together with the auricle, may be moved and displaced in every desirable direction over the temporal bone.

Howarth and Bateman (28) describe the advantage

of the above approach and go on to say not only does the triangular window made in the posterior superior meatal wall give an excellent view of the region on the mastoid antrum, but also the extension of the initial incision forwards and upwards into the soft tissues in the gap between the crest of the heliz and that of the tragus--a gap at which the continuity of the cartilaginous ring is broken--vastly increases the possibility of mobilizing the soft tissues on the underlying bone, and this mobility can still further be increased by understripping the hole of the pinna itself.

In acute cases no packing whatever is required, and perfect drainage from all parts of the bony cavity in the mastoid is assured. The cavity granulates quickly, the triangular gap in the posterior meatal wall, when filled with granulation tissue, is soon covered with epithelium, and the normal contour of the meatus is restored. This absence of packing and dressing is most satisfactory from the patient's point of view, and the inspection of the cavity by the surgeon, should it be necessary, is a simple and painless procedure. Complete healing usually takes place in a month.

XIII Complications

In taking up the complications of acute mastoiditis, the writer will list the possible complications along with a brief diagnosis

In 300 cases of acute mastoiditis operated upon by Krentz and Wetter (40) at Henry Ford Hospital, 48 of the 300 or 16 per cent developed complications. Of the 16 per cent 7 per cent developed persistant otorrhea; 4 per cent lateral sinus thrombosis; 3 per cent meningitis 1 per cent brain abscess and 1 per cent septicemia.

The complications of acute mastoiditis may be listed as;

1. Perisinous abscess
2. Lateral sinus thrombosis
3. Organismal meningitis
4. Serous meningitis
5. Septicemia
6. Extradural abscess
7. Petrositis
8. Bezold's abscess
9. Infectious facial nerve paralysis.

The bacteriology of the complications is given

on the following chart, page 79, which is taken from McCaskey, Sims and Estlick (45).

Petrositis.--Signs, symptoms and diagnosis. Kopetzky (34) gives the following symptoms.

1. Pain in and about the eye-especially during the night.

2. Continuous aural discharge during the period of pain.

3. Low grade sepsis.

4. Onset of suppurative apicitis, usually late--an average of one month after a primary operation upon the mastoid.

5. Palsy of the sixth nerve (not a definite part of the picture).

Kopetzky (34) stated that of laboratory data obtainable, the roentgenograms were the only real aid in the diagnosis of petrositis.

Eagleton (18) concluded from his studies that infection within the petrous apex has a tendency to spontaneous subsidence, efficient drainage of the associated mastoid suppuration being all that is necessary to effect a cure.

Epidural abscess.--Hallberg, Williams and Craig (23) cite a case of a boy of 16 years that complained

	Riley Hosp. Upto 16 yrs.	16 years of age or over	Total	Percentage	Bacteriology of the complications									
					Revisionous abscess	Lateral sinus Thrombosis	Organismal meningitis	Rhasists	Extra dural abscess	Serous meningitis	Bezold's abscess	Infection facial nerve paralysis	Septicemia	
Number of mastoid cultures obtained	182	20	202	100	24	14	17	5	3	3	2	2	21	
Streptococci, undifferentiated	5	0	5	2.4	1		3						2	
Streptococcus hemolyticus	131	12	143	70.3	19	12	4		2		1	1	14	
Streptococcus viridans	7	2	9	4.4							1		2	
Streptococci and staphylococci, undifferentiated	3	2	5	2.4				2						
Total				80										
Staphylococci, undifferentiated	4	0	4	2	1	1	1			1			1	
Staphylococcus aureus	10	0	10	5	1			1		1				
Staphylococcus albus	1	1	2	1										
Staphylococcus hemolyticus with streptococcus non-hemolyticus	4	0	4	2										
Total				10										
Pneumococci, undifferentiated	6	2	8	4	1		3	1					1	
Pneumococcus, type II	1	0	1	.5										
Pneumococcus, type III	1	0	1	.5										
Pneumococcus, type V	1	0	1	.5										
Pneumococcus, type VI	1	0	1	.5										
Total				6										
Hemophilus, influenzae	2	0	2	1			3					1		
Bacilli, undifferentiated gram positive form	2	0	2	1	1			1		1				
Bacilli, gram - Proteus group	0	1	1	.5			1		1					
Mycobacterium tuberculosis	1	0	1	.5			1							
Escherichia coli	1	0	1	.5			1						1	
Diphtheroids, undifferentiated	1	o	1	.5		1								
Total				4										

of motor aphasia and right hemiplegia. A month before the symptoms developed the boy had had measles.

The patient had ear-ache in the left ear 13 days prior to admission. Spontaneous rupture of the membrana tympani occurred 10 hours after the onset of the pain. 9 days later he presented pre-auricular swelling, increased temperature and leukocytosis. With the aid of roentgenograms, the diagnosis of epidural abscess was made. At operation the abscess was found and drained. The patient recovered the use of the right arm and leg movements.

The epidural abscess is associated with mastoiditis and is one of the more complications.

Suppurative meningitis.--Dwyer (16) says that the removal of the focus of infection is of prime importance in the treatment of suppurative meningitis. The surgery of this type is the removal of the foci. The diagnosis of the meningitis is made upon the examination of the spinal fluid. This is the complication that has been treated the most successful with the sulfonamide group, especially sulfanilamide.

Brain abscess.--Davidoff (13) waits until the

abscess is encapsulated until surgery is pe formed for the relief of symptoms. This may take from 4 to 14 days.

Brain abscesses may be classified into:

(1) Temporosphenoidal abscess

(2) Cerebellar abscess

(3) Frontal abscess

A good diagnostic point, but it may be absent in some cases, is choking of the optic disks.

Brain abscess. Differential diagnosis. Ersner (19). Brain abscess must be differentiated from brain tumor, epidural abscess, sinus thrombosis, circumscribed serous meningitis and suppurative meningitis.

Brain tumor--Tumor of the brain generally runs a slower course. There is no fever, no history of suppurative otitis media, or of leukocytosis, not is there evidence of an aseptic meningitis.

Sinus thrombosis--This is characterized by high, remitting fever, chills, positive blood culture, and positive Tobey-Ayer test. At times lateral sinus thrombosis and brain abscess may occur coincidently.

Epidural abscess--Epidural abscess, occuring in cases of acute or chronic suppurative otitis media,

XIV Summary and Conclusions

In discussing either acute mastoiditis or otitis media the two subjects must be taken together.

There are two distinct types of mastoiditis; the coalescent type and the hemorrhagic type. These are both clinically and pathologically distinct entities.

The pre-disposing factors have been taken up in detail and it was seen that age, climate, debilitating diseases, the type of bone, the virulence of the micro-organism and the type of organism were the main factors to be considered in the production of acute mastoiditis.

The signs, symptoms and diagnosis are not definite in some instances but in most cases the history of pain in the mastoid region, together with the discharge, reduction in hearing, sagging in the posterior superior canal wall and the general symptoms such as physical unrest, lassitude and anorexia, give a clear picture as to the nature of the disease.

The roentgenograms are of definite value in the diagnosis of acute mastoiditis. There is some doubt as to the advisability of x-ray therapy in cases of surgical mastoid cases but it is universally accepted that surgery is to be instituted in cases where the

cell walls have been broken down.

The use of sulfanilamide has proved of great value in mastoid disease but as yet it is too early to formulate any definite opinions as to the use of the drug. When mastoiditis is seen early the drug is indicated, but it is to be used in conjunction with surgery if the disease is seen late.

The surgery of the mastoid has become a standardized procedure and the operators have each modified the procedure to fit their own particular needs.

The complications which follow acute mastoiditis depend largely upon the skill of the operator and the after care given to the patient. If complications do ensue, they should be diagnosed immediately and be given the proper care.

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